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DISEASES OF THE NERVOUS SYSTEM

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DISEASES of the NERVOUS SYSTEM

(PUBLISHED MONTHLY)

A PRACTICAL JOURNAL ON PSYCHIATRY AND NEUROLOGY

EDITORIAL COMMENT

Another Contribution From Neurosurgery

At one time neurology was a rather exact science concerned chiefly with the interpretation of symptoms and signs. Upon this basis the lesion was located, the cause determined, and the condition classified, but the neurologist was generally unable to proceed further because the means for treatment were limited to only a few disorders.

Time and discoveries in chemical, biological or bacteriological, and physical realms have gradually reduced the number of conditions that did elicit a bad prognosis. For instance, neurosurgery has contributed many new techniques to the treatment of diseases of the nervous system. Such advances were especially noticeable in the years following the first World War. Cushing's outstanding work with tumors and other special neurosurgical techniques and the introduction of pneumo-encephalography and other contrast media are but a few examples. The advent of the electrocautery and the ever-increasing achievements of medical science have permitted skilled technicians to devise many valuable methods and operations which are too numerous for special mention.

From time to time the Journal has printed various articles written by neurosurgeons illustrating the results of these efforts in the field of neurology. The neurosurgeon has even invaded the domain of psychiatry. Certain chronic functional mental disorders have benefited from an operation called Prefrontal Lobotomy as discussed by Freeman and Watts in the January issue of the Journal. Numerous papers on this subject have appeared in other medical journals and more will surely follow.

Dr. Bucy's paper in this issue about the operative treatment of choreo-athetosis is a further clarification of his previous work about patients who suffer from abnormal movements of their body musculature. In past years the outlook for these patients was hopeless. The pathology and physiology of these purposeless movements is still in question, but it is fairly well accepted that some disease process of the extra-pyramidal pathways causes the abnormal movements. Apparently the cortical center for this system is Brodmann's area six situated anterior to the so-called pyramidal "motor area" (area four). From here the fibers descend to lower centers of the brain and thence down the anterior part of the spinal cord, to eventually reach the anterior horn cells. If this is accurate, it should explain the failure of posterior root sections, for subsequent operative procedures seemed to utilize this knowledge. Putnam[1-2] has been successful in relieving some athetosis by section of the extra-pyramidal tract in the spinal cord. Bucy and Buchanan[3] extirpated a portion of the precentral gyrus in an earlier operation, but based on further studies, Bucy and his coworkers[4] decided to remove a portion of area six of the cortex opposite to the involved limb. This work was confined to cases of unilateral athetosis, but Klemme[5] has operated on both cortical extra-pyramidal areas in the presence of bilateral athetoid movements. The results in both chordotomy and cortical extirpation have been moderately successful, though in a few cases the latter operation has been followed by the development of convulsive seizures. Putnam believes the future operation of choice will depend upon the ultimate success, freedom from complications, and the extent of mortality statistics in each type. All of this work promises to be of much value in the treatment of those conditions of the basal ganglia and the associated extra-pyramidal system, which do not respond to drug therapy.

Hamilton Ford, M.D.
Galveston, Texas

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Alcoholic Avitaminosis*

ABRAHAM MYERSON, M.D., LEO ALEXANDER, M.D., *and*
MERRILL MOORE, M.D.
Boston, Massachusetts

Dr. Myerson is an Associate Editor of the "American Journal of Alcoholism," and a member of the Research Council on Problems of Alcohol. Also, he holds professorships at Harvard Medical School and Tufts College Medical School; consultancies in Neurology or Psychiatry, or both, at six hospitals, and the chairmanship of the State Research Committee of the Department of Mental Health for the Commonwealth of Massachusetts. Dr. Myerson is fifty-nine years old and has worked in the fields of neurology and psychiatry, and writing. The last field of endeavor has produced seven books and an innumerable quantity of magazine articles.

Dr. Alexander received his M.D. degree from the University of Vienna in 1929. Following internships and residentships and teaching positions in Vienna and Frankfurt, he was Lecturer in Neurology and Psychiatry at the Peiping Union Medical College in China during 1933. In 1934 he came to the Worcester State Hospital where he was Clinical Assistant. Following this, he was Junior Visiting Neurologist and Neuropathologist at the Boston City Hospital, Research Associate at the Boston State Hospital, Teaching Assistant and Instructor in Neurology at Harvard, Visiting Psychiatrist at the Washingtonian Hospital and Associate Examiner of the American Board of Psychiatry and Neurology. At present, Dr. Alexander is Associate Professor of Neuropsychiatry at Duke Hospital and Medical School.

Dr. Moore was graduated from Vanderbilt University School of Medicine in 1924. He is an Associate in Psychiatry, Department of Diseases of the Nervous System, Harvard Medical School; Visiting Psychiatrist, Neurological Unit of the Boston City Hospital; and Medical Director of the Washingtonian Hospital, Boston. Dr. Moore is a Member of the American Neurological Association, the American Psychiatric Association, the Massachusetts Psychiatric Society, the New England Psychiatric Society and the Boston Society for Psychiatry and Neurology.

The ingestion of alcohol and its pathological manifestations range from that simple and good-natured drinking which elicits the smile and jest of friends to that chronic excess which may lead to insanity and death due to diffuse brain damage.

The problem of the excessive use of alcohol, called alcoholism, is deeply rooted in the customs of mankind because of the circumstances under which mankind lives and the effect of alcohol upon the feelings, egoism, and the badgered personalities of mankind. Alcohol enables man to effect a transition from reality into something that is temporarily more pleasant; it affords him a seeming amelioration of his hard luck and, in addition, it has become part of the symbolism of the exuberant, joyous occasion. There has been no civilization, no matter how primitive or ancient, that has not used alcohol in one form or another, or some other drug, to release sociability, to escape frustration and psychic pain, and to drown for the moment anxiety and psychic distress.

For good or ill, alcohol seems to be as firmly a part of the life of mankind as his industrial and sexual habits are, and it must be reckoned with in a realistic manner. That alcoholism cannot be abolished by sweeping legislation was sufficiently demonstrated by the recent social experiment of 1919-1933. Even the dissemination of the knowledge that what alcohol does in temporarily relieving the frustrated human being from his psychic distress is paid for exorbitantly in one form or another has not sufficiently deterred people from drinking. Though measures of legislation and education, particularly the application of the provisions of the Federal Food, Drug and Cosmetic Act to the sale and advertising of alcoholic beverages,[1] may finally be effective in deterring men from drinking to excess and from drinking particularly unwholesome types of alcoholic beverages, this is necessarily a slow process, if destined to be accomplished at all. We wish, therefore, to offer an additional practical plan based on certain achievements of medicine in the past generation and utilizing the modern methods of propaganda. These achievements have finally provided pertinent data concerning one of the particularly damaging mechanisms of the harm which alcoholism does to the human organism, namely the pathogenic mechanism of those organic lesions which are caused only indirectly by the ingestion of alcoholic beverages. These medical facts now indicate very illuminatingly what practical points in the prevention of these damages need to be considered. These facts need the methods of wide dissemination which are now part of modern civilization.

* From the Division of Psychiatric Research, Boston State Hospital, aided by grants from the Commonwealth of Massachusetts and the Rockefeller Foundation; the Washingtonian Hospital; the Neurological Unit, Boston City Hospital and the Department of Nervous and Mental Diseases, Harvard Medical School.
Read at the ninety-fourth annual meeting of the American Psychiatric Association, held in San Francisco, June 6-10, 1938.

Clinical Manifestations

Mental disturbances occur in the course of acute ingestion of alcohol, known as pathologic intoxication, but in importance these are far outweighed by those syndromes which follow chronic ingestion of alcoholic beverages. These clinical syndromes of damage to the nervous system due to chronic abuse of alcohol usually do not appear before five years of such abuse, according to a consensus of opinion of many authors and according to our own experience, though occasionally they may appear earlier.[2] The disturbances of the nervous system following chronic alcoholism are: (1) polyneuritis, (2) the alcoholic psychoses, and (3) the alcoholic encephalopathies and encephalo-myelopathies. As to the psychoses, in delirium tremens, in cases of acute alcoholic hallucinosis and in Korsakoff's psychosis, the etiology is, without doubt, purely exogenous while in alcoholic paranoid psychosis and in chronic alcoholic hallucinosis, there seems to be a constitutional predisposition.

The pathogenic mechanism by which alcohol damages the nervous system has recently been subjected to a great deal of research and discussion. The general trend of interpretation is away from the older assumption of direct toxic action on nerve tissue by alcohol itself and more in favor of metabolic, more specifically avitaminotic, disturbances inaugurated by the chronic ingestion of alcohol. This alcoholic avitaminosis is due to three factors:[3,4,5] (1) neglect of proper diet by chronic addicts of alcohol; (2) incomplete absorption of vitamins, if taken, due to gastritis, and intestinal and liver changes produced by alcoholism; and (3) increased vitamin requirement due to the high caloric value of alcohol. This third factor might make a normal balanced diet inadequate for the alcoholic person, even in the presence of normal resorptive functions. However, absolute proof of the avitaminotic causation of alcoholic nervous system damage in man has only been definitely established for polyneuritis.[6,7,8,9,10,11]

There is a great deal of suggestive evi-

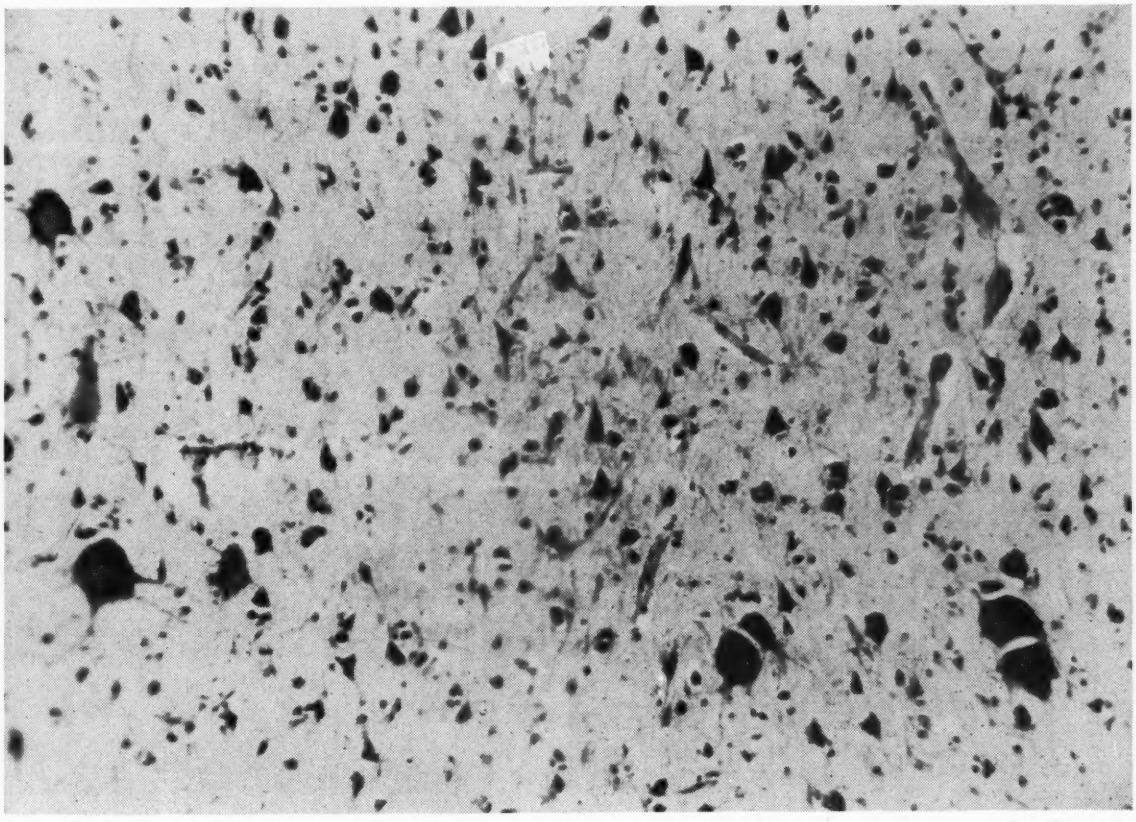

Fig. 1 (Case 1). Central neuronitis (Pellagral ganglion cell disease). The fifth layer of area four of the cerebral cortex in a case of chronic alcoholism with Korsakoff's psychosis and pellagra. Male aged 53. Note extreme swelling of large motor ganglion cells. Nissl stain. Photomicrograph x 150.

dence that Korsakoff's psychosis may be due essentially to deficiency factors, especially if it be complicated by pellagra lesions,[12] or by nicotinic acid deficiency encephalopathy.[13] This clinical evidence receives further support from the fact that a full-fledged Korsakoff's syndrome may occasionally develop in deficiency states due to gastro-intestinal diseases and other systemic disturbances, without the presence of alcoholism.[14] However, much less clear is the situation in delirium tremens, for instance, which has never yet been found fully duplicated in any other toxic or metabolic disease except chronic alcoholism. Although certain acute hallucinatory states occurring in other toxic and febrile conditions (such as cocainism, morphine poisoning, delirium with childbed fever, etc.) may occasionally be reminiscent of delirium tremens, there is still sufficient clinical dissimilarity to allow for a differential diagnosis to be made easily, even without knowledge of the history of the case. However, these uncertainties should not inhibit one from searching further for the possible avitaminotic factors, even in such a specific alcoholic disturbance as delirium tremens, because specific syndromes may be brought about by imbalance in the degree of deficiency of the various vitamins rather than by the presence or absence of any one vitamin alone. Such dynamic and possible quantitative relationships have been suggested by the recent experimental work of Alexander, Pijoan and Myerson.[15] This leads us into the field of pathology where we are much better able than in the clinical field to show that all the pathological changes found in the nervous systems of people suffering from chronic alcoholism can be reproduced by disturbances of the vitamin metabolism.

Pathology and Pathogenesis

According to the general opinion at the present time, the main organic damage that alcohol inflicts on the human nervous system is due to avitaminosis rather than to primary toxic injury from alcohol itself.

Pathologically, two groups of manifestations stand out:

1) A syndrome characterized by central and peripheral neuronitis[16] with swelling of ganglion cells (Figs. 1, 2, 3) predominantly in the motor and frontal cortex and the motor horns of the spinal cord, and with degeneration of nerve fibers, predominantly of the myelin sheaths (Fig. 4) in the peripheral and central nervous systems. This syndrome can be reproduced in animals with Vitamin B deficiency and resembles the clinical syndromes commonly found in persons suffering from beri-beri[17] or pellagra.[18] One of us[19] has recently subdivided the neuronitis syndrome into three distinct sub-types: (a) neuronitis predominantly of *peripheral nerve fibers,* a syndrome identical with beri-beri neuritis and due to deficiency of the B_1 group of the vitamin B complex; (b) neuronitis predominantly of *nerve cells,* especially of the motor cells of the precentral cortex and of the anterior horns of the spinal cord, a syndrome identical with the *central neuritis* seen in pellagra and due to deficiency of nicotinic acid; (c) neuronitis predominantly of the *long tracts* within the least well vascularized parts of the spinal cord, a syndrome identical with the *combined tract degeneration* seen in certain anemias, particularly pernicious anemia, and probably due to deficiency of intrinsic or extrinsic anti-

Fig. 2 (Case 1). One of the diseased cortical ganglion cells, shown in Fig. 1, at higher power. Note extreme swelling of the diseased ganglion cell, flattening and displacement of the nucleus to the right cell margin, chromatolysis and homogenization of most of cell body with only a few remaining Nissl bodies near the apical dendrite and at right margin of cytoplasm, especially adjacent to nucleus. Nissl stain. Photomicrograph, oil immersion, x 1750.

anemic factor, the chemical identity of which has not yet been ascertained. In alcoholic patients this latter type of deficiency is rare.

2) A syndrome characterized by many small foci of angio-degeneration and subacute necrosis of the parenchyma (Fig. 5) with (Fig. 6) or without (Fig. 5) petechial hemorrhages predominantly around the ventricular system, especially the basal part of the third ventricle, about the Sylvian aqueduct and at the floor of the fourth ventricle, first described by Wernicke[20] as polioencephalitis hemorrhagica superior and inferior. Although this syndrome has been found in cases of scurvy in alcoholics as well as in non-alcoholics (by one of us[15c]), and although we[21] and Wortis, Wortis and Marsh[22] have demonstrated that the Vitamin C level in blood plasma in patients suffering from psychoses due to chronic alcoholism is consistently low, so that Wernicke's encephalitis hemorrhagica might easily have been considered as a cerebral form of scurvy, our further experimental work[15a,b,c] has shown that in laboratory animals (pigeons) Wernicke's disease can be reproduced (Fig. 7) by Vitamin B_1 deficiency, especially if the birds are given an ample supply of one or more of the other vitamins (A, B_2, C, D). This fits quite well with the situation in the alcoholic patient. Alcoholics are usually Vitamin B_1 or nicotinic acid-deficient, but they may

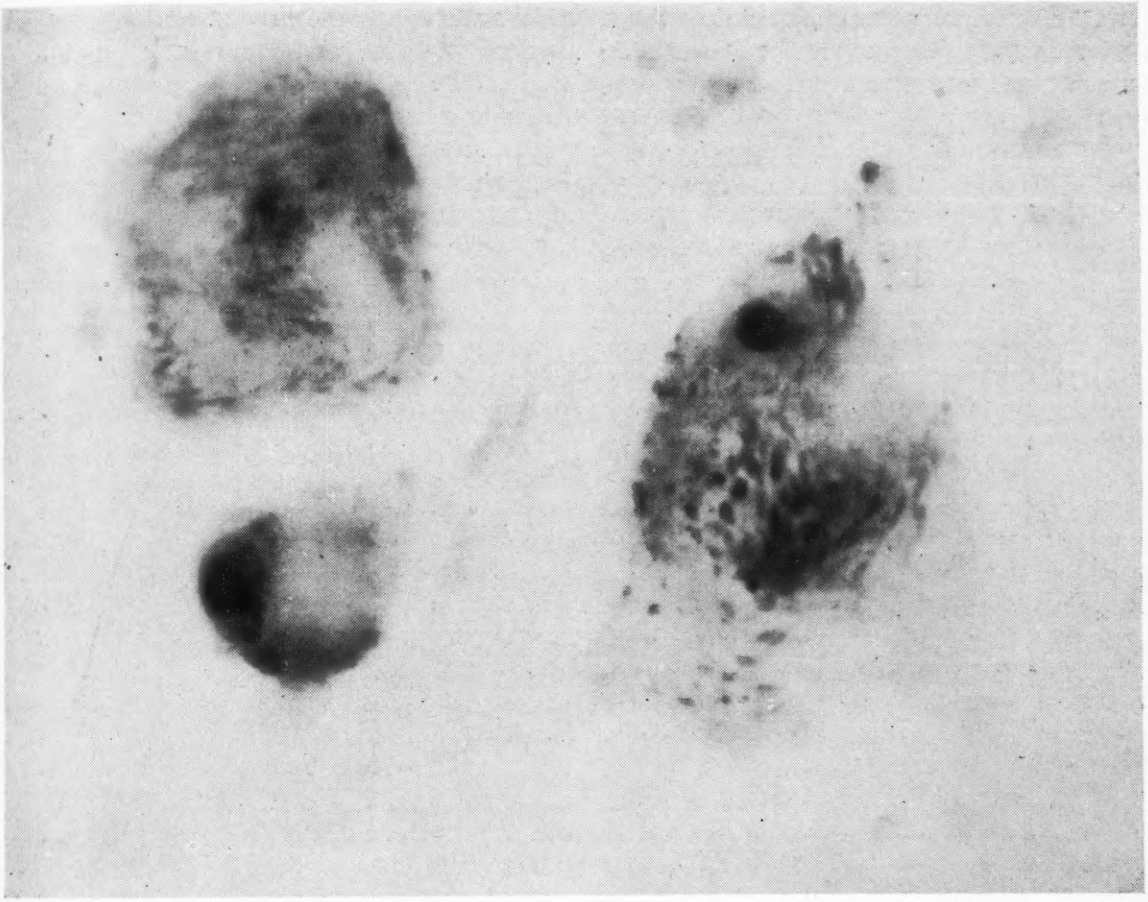

Fig. 3 (Case 1). The various stages of pellagral neuronitis, as illustrated by three neighboring cells from the fifth layer of area four, from a section adjacent to that shown in Figure 1. The cell to the right still retains its original pyramidal shape and normal tigroid bodies are preserved in the center of the cell and about the displaced nucleus; but the left cell border shows bulging with a slight degree of chromatolysis. The nucleus is displaced to the upper left cell margin. There is advanced chromatolysis at the right lower dendrite, at the right lower and the right upper parts of the cell body, and at the apical dendrite. There is beginning chromatolysis in the left lower part of the cell body. The cell shown in the upper left part of the figure shows advanced swelling with rounding of the cell body and diffuse chromatolysis, only a few scattered Nissl bodies being preserved throughout the cytoplasm. The cell in the left lower part of the figure shows advanced swelling with displacement, pyknosis, and flattening of the nucleus, complete chromatolysis in the central, lateral, and upper parts of the cell body with only a few vestiges of degenerated Nissl substance remaining at the base of the cell. Nissl stain. Photomicrograph x 1115.

receive ample supplies of riboflavin. Correspondingly, riboflavin deficiency and a significant degree of anemia[23,24,25] are rare among chronic alcoholic patients. Wernicke's disease cannot be reproduced experimentally by Vitamin C deficiency. Vitamin C deficiency, however, may play a role in the predisposition to subdural hematoma which is more commonly encountered in alcoholic as well as in scorbutic patients[21,26] than can be explained by the higher incidence of trauma alone.

Conclusions

With the establishment of vitamin deficiency as the immediate requisite of damage to the brain in chronic alcoholism, there comes the question, what steps shall be taken to prevent this damage. Obviously, nothing should be done to encourage chronic alcoholism. On the other hand, while we are waiting for the social millenium in which men will use alcohol in moderation and with due regard for their own social welfare, it is definitely possible to start a social propaganda by which the facts shall come to the attention of the chronic alcoholic himself.

Society has built up in its industrial advertising a fine mechanism for bringing to the attention of the consumer of alcohol the necessity for ample ingestion of food, to be supplemented if necessary by vitamin tablets, while drinking. Proper application of the provisions of the Federal Food, Drug and Cosmetic Act[1] would be helpful in this direction. The great drug houses which manufacture the vitamins should be enlisted in this campaign, since propaganda carried out by medical men alone does not have the force and the scope necessary for a successful program of this nature. In every bar room, in every café, in every cocktail lounge, it should be brought to the attention by appropriate legends that the drinker must eat well, and

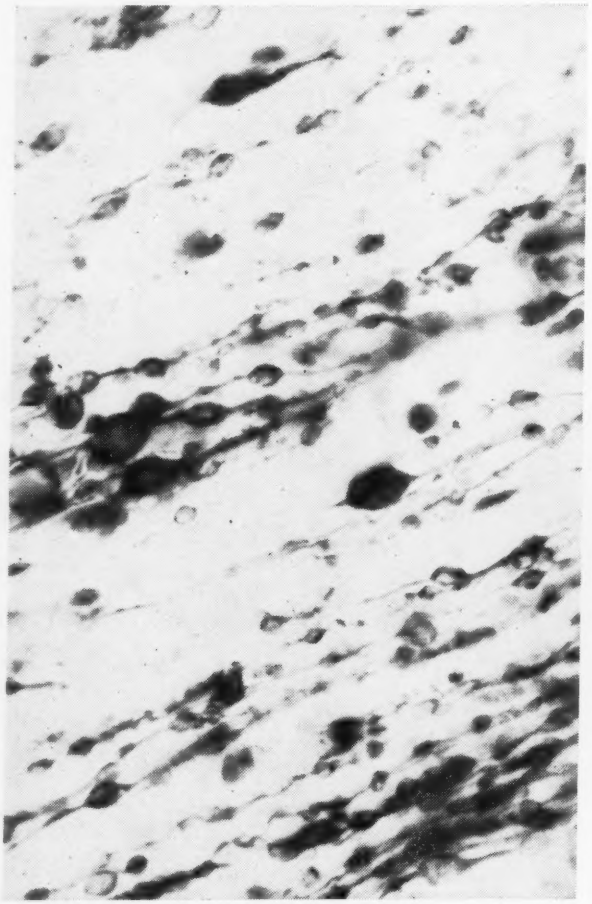

Fig. 4 (Case 2). Neuritis of femoral nerve. Incipient delirium tremens and polyneuritis in a chronic alcoholic female patient, aged 40. Stain, Weigert-Pal. Microphotograph. Enlargement x 1300. Note varicose swelling (beading and ballooning), thinning and complete destruction of myelin sheaths.

Fig. 5 (Case 3). Wernicke's disease in a case of chronic alcoholism with acute Korsakoff's psychosis. Male aged 50. A lesion from the paraventricular nucleus of the thalamus showing subacute necrosis of the parenchyma and angiodegeneration but no hemorrhages. The adjacent normal tissue is shown in the lower part of the figure. Note varicose dilatation of vessels of all sizes within the lesion with irregular tortuosity of the degenerated walls which show delamination of the remaining collagenous strands and hyaline degeneration of intervening lemellae. Masson's trichrome stain. Photomicrograph x 70.

if necessary, add vitamins (particularly vitamin B complex) to prevent serious damage to himself. These vitamins should be conspicuously and immediately accessible either in the form of yeast or in the form of tablets. The man who dispenses the drinks might be put in the role of the dispenser of vitamins as well. The vitamins to be dispensed to chronic alcoholic patients are particularly: Vitamin B_1 (thiamin), nicotinic acid, and ascorbic acid. Though we have no experience as yet as to the minimal protective doses, on a diet of one quart of whiskey daily, we may assume that, pending results of further study, a daily dose of twenty milligrams of each of these substances would be satisfactory.

Fig. 6 (Case 4). Wernicke's disease (Polioencephalitis hemorrhagica) in a case of delirium tremens followed by attacks of alcoholic hallucinosis and terminating in Korsakoff's psychosis in a chronic alcoholic female patient, aged 65. Stain, hematoxylin-eosin. Microphotograph. Enlargement x 8. Note perivascular hemorrhages and subacute necrosis of tissue limited to the mammilary body, which is shown in the lower part of the figure. The remainder of the paramedian region of the hypothalamus and thalamus, which is shown in the upper part of the figure, has remained intact in this case.

This will seem like an encouragement of alcoholism to many, but we seriously doubt this. In fact, it is likely that the addition of vitamins will lessen the urge for the next morning's bracer of alcohol and may, in the long run, lessen the actual amount of drinking. It is the duty of physicians to enlist all agencies that may help to prevent injury and damage to the human being, and in this instance the most important social agencies for the purpose of preventing vitamin damage are those which sell vitamins. The vitamins might also be added to the alcohol itself, although this is at present illegal. Legislation overcoming this difficulty could easily be passed.

It has been shown[27] that the most important factor in alcoholism is the social attitude. So long as the alcoholic is a source of humor rather than disgust, so long as intoxication by alcohol is not viewed in its proper light as a source of injury to the nervous system, so long will men drink to excess. Consequently, the most important way of reducing the damage due to alcoholism is by a far-flung campaign against drinking to excess. This is another story and one not directly within the scope of the present

Fig. 7. Wernicke's disease (polioencephalitis hemorrhagica) in the pigeon, produced experimentally by Vitamin B_1 deficiency, in the presence of ample supply of Vitamin B_2 (riboflavin extracted from yeast and esterified with phosphoric acid, 0.02 mg. daily) and Vitamin C (13 mg. daily). Sagittal section through brain. Photograph x 8. Note perivascular hemorrhages and subacute necrosis of tissue in paraventricular and paramedian nuclei of thalamus and hypothalamus and in periaqueductal region of midbrain. (From material of Alexander, Myerson and Pijoan, 1938).

paper. For the immediate need, the addition of vitamins to the dietary of the alcoholic is primary and pressing.

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Surgical Treatment of Choreo-Athetosis

PAUL C. BUCY, M.D.
Chicago, Illinois

Dr. Bucy is a graduate of the College of Medicine of the State University of Iowa, 1927. He is Associate Professor of Neurology and Neurological Surgery at the University of Illinois College of Medicine. Dr. Bucy is a member of the American Board of Neurological Surgery, has been certified by the American Board of Psychiatry and Neurology and is a member of the American Neurological Association, the American Physiological Society, the Society of Neurological Surgeons and the Harvey Cushing Society.

For many years neurologists and neuropathologists have known that the most common site of pathological alterations in the nervous systems of victims of choreo-athetosis areas is in the basal ganglia, usually in the putamen and caudate nuclei. Less frequently similar abnormal motor states are associated with destructive lesions in the globus pallidus, the thalamus and much more rarely the superior cerebellar peduncle. It is obvious that the involuntary movements of choreo-athetosis could not be produced by these mechanisms which are destroyed. It seems most likely therefore that destruction of these subcortical centers releases some other motor mechanism to abnormal excessive activity. What this other motor mechanism is, remained for years one of the unsolved problems of neurology. Only the late Kinnier Wilson was impressed by the fact that in the early 20th century Sir Victor Horsley had, on at least three occasions, abolished the involuntary movements of choreo-athetosis by extirpating the precentral cerebral cortex, and that Wilson himself, like many other neurologists, had observed that the development of a hemiplegia in a victim of choreo-athetosis abolishes these involuntary movements. Parkinson, and later others, had noted the same thing in connection with the tremor of paralysis agitans. Wilson therefore logically concluded that the nervous impulses responsible for the involuntary movements of choreo-athetosis arise from the precentral cerebral cortex, descended from there via the pyramidal tract to the anterior horn cells of the spinal cord and are transmitted thence to the skeletal muscles. Wilson's views of this subject, however, received little attention and it was generally held that the basal ganglia were the only cerebral structures concerned with these involuntary movements. However, during the past decade the correctness of Wilson's hypothesis in general has been established. In collaboration with Buchanan and with Case, I have confirmed Horsley's observation that the involuntary movements of choreo-athetosis, as well as those of hemiballismus, intention tremor and Parkinsonian tremor, are abolished by extirpation of the precentral area of the contra lateral cerebral hemisphere. This fact has been further corroborated by Sachs, Putnam and by numerous other workers whose observations are as yet unpublished. In one minor regard modern views differ from those advanced by Wilson. It is now believed that though the nervous impulses responsible for the involuntary movements of choreo-athetosis arise in the precentral cortex they descend from there, not by way of the pyramidal tract as Wilson thought, but via other efferent fibers which originate there and which have been called the parapyramidal fibers. This concept of the "non-pyramidal" yet cortical origin of the impulses responsible for these involuntary movements was first advanced by Bucy and Buchanan and later supported by observations of Bucy and Case. The confirmation of this concept was supplied by Putman when he demonstrated that the involuntary movements of choreo-athetosis can be materially reduced by section of the anterior columns of the spinal cord, leaving the pyramidal tracts untouched. That the anterior columns, though they may be the most important, are but one of the descending spinal paths conveying parapyramidal impulses responsible for choreo-athetosis is indicated by the fact that not infrequently the marked immediate amelioration produced by anterior cordotomy is not maintained and within a few years the movements in some cases again become severe even though they do not return to their original violence.

The parapyramidal fibers which arise from the smaller ganglion cells in areas 4 and 6 of the precentral cortex subserve a variety of functions. Some of them are capable, either

under the influence of electrical stimulation or spontaneously in the conscious animal, of evoking movement in the skeletal musculature quite independent of the pyramidal tract. Other of these fibers convey impulses of an inhibitory character, and are capable of suppressing activity in the effector cells of the precentral cortex or of suppressing the reflex activity of the subcortical centers responsible for the postural and righting reflexes. Still other activities of the parapyramidal system appear to be the co-ordination and control of the so-called autonomic nervous system, influencing visceral activity, the vasomotor mechanism, etc.

In contrast with the pyramidal fibers, the parapyramidal fibers all terminate in the subcortical nuclei of the brain, such as the pontine nuclei, the nuclei of the reticular formation of the mesencephalon, the substantia nigra, the basal ganglia, etc. From these structures the nervous impulses are conveyed downward by other neurones. As yet we know little of the specific functions of these different fractions of the parapyramidal system. But in one respect, particularly related to the problem of choreo-athetosis, Dusser de Barenne, McCulloch, Garol and Bailey have recently contributed a most useful bit of information. They have shown that fibers arising from two small strips of the precentral cortex, (designated as 8s and 4s) are concerned with the suppresion of the activity of the remainder of the precentral cortex. These impulses descend from areas 4s and 8s to the caudate nuclei and thence travel back to the precentral cortex by way of the globus pallidus and the ventro-lateral nucleus of the thalamus. It thus appears most likely that the lesions in the caudate nucleus, the globus pallidus and the thalamus which release the involuntary movements of choreo-athetosis do so by interference with the transmission of these suppressor impulses from areas 4s and 8s of the precentral cerebral cortex. Similar inhibitory or suppressor mechanisms can be demonstrated in connection with intention tremor and Parkinsonian tremor but they need not concern us here. With this new physiological information the pathology and mechanism of choreo-athetosis becomes clear.

In brief it may be said that choreo-athetosis develops from the interruption of a circular suppressor mechanism which arises from areas 4s and 8s of the precentral cortex and passes by way of the caudate nucleus, globus pallidus and thalamus back to the precentral cortex. Abolition of this suppressor influence releases areas 4 and 6 of the precentral cortex to abnormal activity which is conveyed downward by the parapyramidal fibers to subcortical nuclei and thence principally via the anterior columns of the spinal cord to the anterior horn cells and the skeletal muscles.

On the basis of this physiological understanding of this motor disorder the possibilities for surgical therapy become clear. The neural mechanism giving rise to the involuntary movements may be interrupted either at its point of origin in the precentral cortex or in the anterior columns of the spinal cord. As we are at the moment ignorant of the exact path followed by the impulses passing between these two points any attempt at this time to interrupt this mechanism in its subcortical course to the spinal cord must be regarded as purely experimental. Destruction of the motor mechanism responsible for these movements at the cortical and at the spinal levels both have their advantages and limitations.

Section of the anterior columns of the spinal cord for the relief of the involuntary movements of the choreo-athetosis is an operation devised and advocated by Putnam. Its great advantage lies in the fact that the operation does not produce any demonstrable disturbance of the voluntary control of the skeletal musculature or increase the disturbance of voluntary muscular control from which these patients so often suffer and that it is applicable to bilateral as well as unilateral disorders. However, due apparently to the fact that the anterior columns of the spinal cord convey only part of the descending fibers responsible for these involuntary movements, the operation of anterior cordotomy does not completely abolish the involuntary movements although it usually reduces their intensity appreciably. Furthermore, in many instances the initial improvement is not fully maintained and after one or more years the involuntary movements again increase in severity returning toward their pre-operative state. It is not to be de-

nied, however, that in many instances a material and much appreciated improvement persists in spite of these facts.

Extirpation of the precentral cortex has the very real disadvantage of producing a definite though not complete paralysis of the contralateral extremities. The amount of voluntary movement which remains after the operation varies from case to case. But the innervation provided by the opposite precentral cortex and the remaining bits of this cortex at the site of the extirpation have in every case provided sufficient voluntary control to enable the patient to use the lower extremity well and the upper extremity for cruder duties. The fact of this unavoidable paresis, however, completely precludes the possibility of a bilateral operation and restricts the usefulness of this procedure to those cases where the involuntary movements are exclusively or predominantly unilateral. In those cases where the choreo-athetosis is associated with a spastic hemiparesis, as it so often is, the paralysis which is secondary to the operation will be a matter of little concern. Likewise in those cases where the involuntary movements are so violent as to render the extremities useless the substitution of some degree of spastic paralysis for the ever present, uncontrollable movements will be welcomed by the patient. In all other cases it behoves the patient and the surgeon to carefully balance the improvements against the disadvantages. In any instance where the patient is dubious about exchanging the involuntary movements for some degree of paralysis, the operation should not be performed.

At first the extirpations were very cautiously made and although the involuntary movements were completely abolished initially they subsequently returned to a limited degree. With inclusion of area 4, the area of the gigantic Betz cells, in the extirpation and extending the limits of the incision widely so as to insure inclusion of all the representation of the involved extremities it has become obvious that the more generous extirpations are productive of the best and most lasting results. It is also noteworthy that the same operation is equally effective in abolishing both intention tremor and tremor at rest.

25 E. Washington St.

Some Psychiatric Aspects of Total War

W. LINDESAY NEUSTATTER, M.B., M.R.C.P.*
London, England

Dr. Neustatter is a member of the Royal College of Physicians of London. He is Physician in Charge of the Department of Psychological Medicine, Queen Mary's Hospital for the East End, London; late Research Assistant in Clinical Psychiatry, Guy's Hospital; Psychotherapist to Out-Patients, the Maudsley Hospital; Commonwealth Fellow in Child Psychiatry and Assistant Medical Officer, the State (Mental) Hospital, Worcester, Massachusetts. At present, Dr. Neustatter is engaged in war work in England.

Whereas the man at the front is a whole-time soldier, the civilian is in the curious position of being a part-time soldier. A large portion of the time he is trying to lead a normal life and yet for a short while, may find himself combining this with being the object of war-like attentions. Thus I have had a game of cricket interrupted by bombs falling on a neighbouring hospital; I have been playing squash when the whole squash court has shaken from the reverberation of a bomb; and I have played hockey with an aerial battle raging overhead. I do not want to give the impression that my sole activity in the war is playing games, but it is an apt illustration of the curious circumstances which arise from total war.

The soldier who is at the front has as his *entire* job waging war—he is not expected to carry on normal civilian life in the day, while fighting an action at night, but the A. R. P. warden or the auxiliary fireman often does so. And while the A. R. P. worker on duty can also feel that, like the soldier, he is taking risks in the course of his duty, it is annoying to feel the necessity for running risks in the course of taking one's pleasure. To be bombed in the local cinema or blasted

* Physician in Charge of Psychological Medicine, Queen Mary's Hospital for the East End of London; Psychiatric Specialist in Charge of a Department in Kent in a War-time Medical Service.

out of the local pub has not even the consolation of dying a hero's death!

Direct Psychological Effects of Air Raids on Civilians

Safety, lack of sleep and destruction of homes are three major aspects of this problem.

Safety—It might be said that the desire for safety is interwoven with a desire for comfort and the necessity of pursuing a normal existence. For the moment, I am ignoring the question of carrying on vital work, which is really a different problem, comparable to members of the Services obtaining such security as is compatible with the performance of their duties. The necessity for carrying on normal everyday life is rather different. To get one's hair cut or to buy cigarettes is not a national necessity (though the need for doing household shopping is nearer), yet from the point of view of general morale, it is very important that life should go on as normally as possible. At the beginning of air raids, even in little-raided districts, shops closed and people took shelter, but now—added to by the fact that there is little day-time aerial activity—life proceeds as normal.

Comfort—At night this is very important to many, almost surprisingly so. To some persons, discomfort appears to be a worse alternative than the risk of injury; and, except in the very worst raided districts, people can be found sleeping on upper floors. There are, however, habitual tube dwellers who, apart from those whose homes give little protection, find that the safety of the tube counterbalances their discomfort.

Shelters—The attitude adopted by people towards shelters is an extremely important one. The *feeling* of security which is afforded is very important in itself and it is often produced, or absent, on quite irrational grounds; yet provided the feeling is there, it is obviously beneficial in ensuring rest and diminishing nervous strain. "Mass Observation" reports that the psychology of sheltering is primarily an emotional one. Thus, Anderson shelters were considered to be part of a home, especially by women, who sometimes took a personal pride in them. However, brick shelters were objected to on sometimes absurd ground, because some people thought they would act as targets. One woman disliked them because "they are so inflammable."

Tubes are considered the safest, and trench shelters are particularly popular, not only on grounds of safety (there is actually a danger of flooding in some) but because people feel away from the raid, are protected from noise and, in some cases, escape the feeling that a bomb is being aimed directly at them. A shelter marshal commented on the fact that the public does not sufficiently realize that the vast number of shelters are no protection against direct hits, but that they are blast and splinter-proof, which materially decreases the danger. The people whose nerves are tried the most are those who are neither fatalistic nor have tube shelters. A good example of securing a feeling of safety with no rational foundation whatever but only the idea of safety associated with the tubes, is instanced by a family who regularly sheltered at the top of the stairs leading down to the tube. They had no protection overhead other than a glass roof, but they would not on any account stay at home, as it was not safe!

Shortage of Sleep—Shortage of sleep is obviously a serious problem owing to its deleterious effect on working capacity, and the nervousness and irritability which follow in its wake. The following table produced by "Mass Observation" is, therefore, both interesting and heartening:

On the 15th of September 1940, 19 per cent of males and 37 per cent of females had no sleep at all. Forty-three per cent of males and 70 per cent of females had less than 4 hours sleep. During November, 4 per cent of males and 8 per cent of females was the highest number without sleep; often this figure dropped to 0. Nine per cent of males and 25 per cent of females was the highest number getting less than 4 hours sleep in November, and at the end of November both these figures dropped to 0. Throughout the table it is noticeable that the percentages of women deprived of sleep is nearly always 10 to 20 per cent greater than that for men.

People who made themselves comfortable slept better than those who did not, and men are reported as using more complicated apparatus to achieve that end than women.

Morale—One of the questions frequently

concerns the role psychology can play in preparing people for the eventuality of war, or in sustaining morale.

This is a particularly difficult question to answer—for not only in England would no one dream of consulting a psychiatrist about it—but it is a question which really leads into politics rather than psychiatry. It is obviously linked up with what the individual considers sufficient grounds for fighting. Psychology might play a role in pointing out the consequences and fallacies in certain arguments; for example (and here I am expressing my own views), it appears to me a fallacious argument that one should only fight where one's so-called vital interests, defined in the narrowest terms, are at stake. If we apply the same reasoning in civilian life that we do in international affairs, we would say that it is a matter of indifference that the house down the street had been burgled, because no one had attacked our house, and that until ours *was* attacked we proposed to do nothing. It might also be possible to point out how perfectly sincere movements—for example, in the cause of peace—can be made use of by those who have no interest in peace, but find such movements convenient for their own ends.

There is one argument I heard put forth during a nine months' stay in the U. S. A. in 1938-39, and that was that to fight for democracy meant that democracy had to be destroyed in the process. Quite frankly, this strikes me as a rationalization. It assumes the impossibility of democracy surviving at all in war, and here I may add an interesting observation on our own democracy. Shortly after Dunkirk, a number of prosecutions for spreading gloom and despondency occurred, and individuals had enormous fines and sometimes imprisonment inflicted on them for making remarks of quite a trivial nature, sometimes in the form of jokes. The prosecutions soon ceased and many of the punishments were reduced, and this occurred as a result of the pressure of public opinion. Some outspoken articles appeared in the press, but the most important factor was a widespread feeling among classes and people of all shades of political opinion, that this sort of thing was "just damned silly." This seems to be one instance at least that democracy in England at any rate has firm foundations.

To return to the question of morale. It is noticeable how much better morale there was at Christmas 1940 compared to the year before. The knowledge that Britain remained alone woke people up and, I think, produced a kind of grim pride: "now it is up to us." Some interesting reactions were observable during the very trying time of last summer when Leopold threw in his hand. I heard the news along with others at a local inn in the village where I was billeted. The reaction was a maniacal one — people behaved as though we had celebrated a victory. Our rapid misfortunes seem so preposterous that they became almost funny. I feel that "maniacal" really describes the reaction better than "hysterical." It was more a feeling of elation, and a pressure of activity, than the alternation of laughter and crying which occurs in an hysterical outburst.

At the time France collapsed many people got drunk and made a lot of noise, but there was not a feeling of despondency, though there was, naturally, considerable apprehension, and the worry of waiting to see where and how soon Germany would strike next. With the desertion of France, I think one could also discern a feeling of relief that "thank God there is no one left who could leave us in the lurch." There was, however, as the result of the nature of the French defection, a feeling expressed among the country people that "if there were any ratting here" it would not be among the common people.

Industrial Efficiency—It is unlikely that American production will require the sudden speeding up which was necessary here after Dunkirk; nevertheless, the reminder of the Industrial Health Research Board that the remedy for fatigue is rest, is a timely one. In a report the Board draws attention to the fact that misguided efforts to stimulate workers to feverish activity in the supposed interest of output, are foolish. Overlong hours and continuous work without intervals for rest are to be avoided. Sunday rest and holidays should be given, and boredom should be alleviated by distractions such as music. (It might be mentioned in passing that very successful lunch-time concerts are regularly held for war time workers. J. B. Priestley in a recent broadcast testified to their beneficial influence.) They conclude, with the impor-

tant warning to managements that "discontent can be the thief of 'output'." Vernon has also summarized some of the findings of the last war, and the necessity for employing this knowledge in arranging shifts of work. For example: alternate weeks proved better than alternate fortnights for night work. A two-shift night-work system is advocated, whilst night work for women is considered inadvisable. In particular, he shows that Sunday labor was uneconomical in the last war.

I have no first-hand knowledge of problems arising in factories, but my stenographer, Mr. Charles Lane, a reliable observer, who is at present engaged in war work in a factory, comments as follows:

Night Work—"Some workers at first welcome night work as a change from the daily grind (and perhaps there is a certain adventure in having your mid-day meal at half-past two in the morning, with the guns banging away), but as a routine it is a failure. A machine-tool setter in a large factory, who has been working 'nights' for over a year now without a break told me that his operators were turning out work that was 'putrid.' He attributed this to the continuous night work without a break, and the lack of holidays as a result of the 'go-to-it' fever of 1940.

"However, one shift which has proved extremely popular, particularly with women, is the 24-hour shift. In this the hands commence work at 10 a. m. one day and come off at the same time the next, when they are free for 24 hours. A break is made for sleep from 11 p. m. to 4:45 a. m., when breakfast is taken for an hour. Originally the break was from 6:30 p. m. to midnight, but some of the workers were in the habit of not sleeping during that time, but indulging in recreational pursuits instead, with the result that their ability to work subsequent to the break was a trifle impaired and, indeed, in some cases completely obliterated.

"The attraction of the 24-hour shift is the whole day off, and the fact that every other week the worker is free from Saturday morning to Tuesday morning; although, of course, during the other week, he gets Sunday off only, resuming work on the Monday morning.

"Another reason which occurs to me for its popularity is that it is a complete change from the set working routine they have followed for years, particularly as there is no getting up early and scrambling round to clock on by 7:30 or 8 o'clock.

"There has lately been a suggestion that the management might discontinue this shift (for reasons which are no reflection on the workers' output), and there has been much complaint from the women as a result."

Curtailed Lunch Hour—"When the hours of darkness were at the maximum, and air-raids began in the early evening, in order to get the employees off the premises by the time the sirens went, some factories reduced the time of the break for lunch and allowed the employees to cease work for the day correspondingly early. Though the idea was well meant, from the point of view of output it was a failure. In the factory in which I work it meant clocking off, washing (if you could afford that luxury), getting served at the canteen (a fifteen-minute operation at the best of times), 'eating' your lunch, clocking on, and resuming your place in the shop in a sufficiently eupeptic condition to permit of the production of useful work all in half-an-hour. I tried to make the best of a bad job with the sandwiches and a glass of milk, but the diet palled and was certainly not satisfying."

Smoking in Factories—"Although in a large number of wartime factories, where the type of product makes it practicable, smoking is permitted at the benches, some factories—in the interests, I fancy, of a reduced fire insurance premium—still disallow it. This is a short-sighted policy, for when this is the case, at a conservative estimate I should say that one hour per day per smoker is wasted in the lavatories."

The Incidence of Neurosis — Remarkably few cases of war neuroses have to date been admitted to the special hospitals provided exclusively for this purpose. Two of those in the London area subsist largely on Service cases, two more which were temporarily closed during the quiet period never reopened, and I have just seen a report from Liverpool commenting on the very low incidence of cases seen there. Everywhere there has been a complete absence of panic or hysteria.

However, it may be that certain cases of neuroses which should find their way into hospitals do not always do so. There is some

evidence for this. Tom Harrisson, writing in the "British Medical Journal" recently suggested there were quite a number of persons in evacuated districts who were suffering from a form of inert depression and general listlessness with lack of interest, who had not come under medical care. I have seen patients for the Ministry of Pensions (though not of this type) who had never seen a psychiatrist until their claims to a pension were reviewed six months after the bombing, and the Citizen's Advice Bureau has suggested that there are a good number of cases who are "very nervous" among evacuated civilians, which would tend to substantiate Harrisson's statement. On the other hand, as the cases I saw claiming pensions come from all over the Southeast of England, their number, (about 30 in 6 months) is negligible. The comments of the Citizen's Advice Bureau may be a clue to why certain patients never reach a psychiatrist. Nervousness at air-raids, largely shown by apprehension and shaking at the lugubrious sound of the siren, is probably regarded by the victim as a not surprising aftermath to some terrifying experience, rather than a condition requiring medical attention. There is quite likely a good deal of psychological truth in this tacit assumption. In other words, these patients without being aware of it, realize that theirs is a "fear state," a conditioned reflex to the sound of sirens and aeroplanes, rather than an "anxiety state" if we accept the usual definition of anxiety as a reaction to a normally innocuous situation, and fear as a response to a reality situation. And if a 100 lb. bomb does not constitute a reality situation it would be interesting to know what does.

Another reason—although I trust only an occasional one—why patients may not find their way to the psychiatrist is instanced by the conversation of a soldier to whom I gave a lift one day in my car. He was going home to his wife, who was in bed. I asked what was wrong, and he said it was "her nerves after bombing." I suggested that there were "nerve" hospitals for these disorders, specially provided for cases such as hers. But he would not hear of it. He declared that she had seen doctors before, but "they didn't do no good when it was nerves!" Again I fear that to some extent he was right. Anxiety states, or "conditioned fear states," as I suggest they should be called—with the exception of the acute states described later—are very intractable conditions to treat. I have heard Aubrey Lewis express the same view. And, indeed, it is rather hard to know what to say, for example, to someone of an imaginative and apprehensive disposition who has been through a long stretch of raids, suffered from interrupted sleep (an important point), and possibly finally has had his house blown away from him.

But in spite of what I have written here, the amazing thing is not the people who *do* develop anxiety states, but the extraordinarily large number who do not. During the bad winter months, when London had its regular nightly blitz, it was quite incredible how little strain was noticeable in the appearance or demeanor of Londoners. I am not pretending for a moment that people did not mind, but there are the most astounding examples of the adaptability of people to the severest strains. There was no panic. It is also interesting that people exposed to blast, who are not injured but who are brought into hospital pale, shocked, and looking very ill, appear to make a complete recovery, and are fit for discharge in about three days.

The following case histories further illustrate this point:

1) This concerns a single educated woman of 40 who runs a boarding-house in Hampstead. The neighbourhood was one that came off comparatively light in the raids, but she complained bitterly of the intense noise of the barrage, which at first prevented sleep. However, she learned to sleep through this, and confessed with surprise how little worried she was by the raids.*

2) Two men of 65 and 67, the former a taxi-driver, were both sent to a country hospital because they had had their houses bombed, and had suffered from "nervous shock." They appeared rather shaken at first, but both recovered rapidly. The taxi-driver did so in three weeks, after which he insisted on returning to London to drive his

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* Since writing this I have seen an ex-patient, a man of 37, and a highly-strung individual who faints at the sight of blood. He has lived in London throughout the winter, and though having no worse experience than incendiary bombs next door, told me he was surprised at how acclimatised to raids he had become.

taxi. The other was well in a week, and did not have to go back to London. But the surprising thing was that he and his wife lived in Greenwich—a very unhealthy neighborhood—yet although they had been given the chance to evacuate earlier, they had not done anything about it until their house was blown up. "And now," the old man said, "we'll 'ave to, because there ain't no choice." The Cockney is truly a remarkable person.

Clinical Manifestations—Broadly speaking, the disorders met with fall into two categories, viz., acute cases and subacute and chronic conditions. The latter resemble the neuroses seen in peace time. They show the usual clinical picture of anxiety states, hysteria, obsessional states, occasional psychoses, etc. I do not propose to discuss these disorders in detail, however, as my object in this paper is to concentrate only on those aspects of psychological problems which are peculiar to civilians in war.

Subacute and Chronic Conditions

*Anxiety States**—These are far and away the most common conditions. They generally occur, though by no means universally, in those who have previously been subject to anxiety, and (as mentioned) they are apt to be very intractable, responding poorly to any treatment in my experience, other than being removed to a reasonably quiet neighbourhood. Unlike the hysteric they are not "unconsciously attempting to escape," but exhibit anxiety as a direct reaction to stress, though there is always a tendency for a secondary hysterical mechanism to occur.

In those cases which show anxiety with very little outside stress, I believe the Freudian theory is true, namely that their anxiety is brought about by repressed aggressive tendencies, the horrors of war being both too near their unconscious sadistic wishes and their fear of punishment, for the patient to be able to tolerate them. I do not know how psychoanalytic therapy would help in such cases; moreover, there is not time for such elaborate methods in war, and as the course of treatment might outlast even the most pessimistic expectations of the duration of hostilities, it might be hard to check up the results!

Anxiety States and Their Treatment—Partly in order to try and prevent hysterical manifestations, the special hospitals for neuroses have not been put in particularly quiet areas, with the express object of avoiding the difficulties of getting patients back into the "war zone." Indeed, in order to try and get patients reacclimatised to war conditions, attempts have been made at some hospitals to "decondition" patients by producing the sound of explosions, and by turning an aeroplane engine on unexpectedly. I consider the theory behind this to be unsound. To decondition a conditioned reflex surely means that the conditioning stimulus must no longer be followed by the situation producing the psychological reflex. Now although the fear produced by the sound of an aeroplane engine, or an explosion, if not followed by danger might become innocuous if heard repeatedly *after* the war, during the war the patient is bound to realize that he will have to return to danger later on, and therefore the first stimulus does not really appear as innocuous. I believe this method is attended by dubious success, and I heard that in one hospital, where a sound-film of Dunkirk was used for this purpose, it had to be discontinued because of its deleterious effect on the nerves of the medical staff!

Other psychotherapeutic methods used are the usual shorter ones of discussion, persuasion, and suggestion, combined with a regular routine of exercises and drill. Sociological factors may play an important role in perpetuating these conditions. Concern about relatives in bombed areas, or worry at the loss of homes, may hold up recovery, and the services of some of the special social agencies set up to deal with these problems, in securing information or giving advice, may be invaluable in promoting betterment.

Obsessional states—These are exceedingly rare. I have not seen one, and the war appears to act chiefly as a precipitant. They need treatment along ordinary lines.

Hysteria—This appears to be remarkably uncommon—less than was expected. Possibly, as Stephen Taylor suggests, it is that patients realize that in a psychiatrically-minded age they will be more likely to receive sympathetic attention if they complain of depres-

* Although I have suggested the term "fear state" as a better one, it is simpler to conform to the accepted nomenclature.

sion rather than a paralysed leg.* The few cases I have seen have generally been in connection with compensation.

For example, a woman of 67, of sound personality, who was a chiropodist in Brighton, had her home blown away and was thrown against a wall by the blast. She was slightly concussed, but did not seek medical aid for some weeks, when she complained of headache, nervousness and tremor. I saw her six months after the bombing. She was very emotional, and had an obviously hysterical tremor. The tremor prevented her practising chiropody, and consequently she was bereft of a livelihood. It also transpired that the neighbourhood she was in was frequently bombed (she remarked that the Germans did not appear to bomb any of the large hotels but concentrated on the poor neighbourhoods). There was consequently no opportunity of setting up in business again. The motivation for her hysteria was clearly due, in large part, to the fact that her clientele had scattered as a result of the bombing of the neighbourhood, and she agreed feelingly that the medicine she needed was some security for the future, which we tried to find for her. Even instigating steps in this direction, with a rest in hospital, brought about much improvement.

Most of the other cases I have seen were similar, the motive arising from financial insecurity. And I would like to stress, in view of the unsympathetic attitude adopted by some authorities towards all compensation cases, that in these cases the motive was neither dishonesty nor gold-digging; it was a perfectly comprehensible anxiety about the future, in some instances possibly combined with an underlying, if undefined, feeling that their loss might reasonably be distributed over the community as a whole.

Depression—When this occurs, it is usually associated with anxiety. As remarked earlier, T. Harrisson has commented upon evacuees who are suffering from an inert and depressed listlessness. I have no first-hand experience from which to write on this topic. In contrast to this, I have heard of several cases of elderly persons who before the war had been depressed, hypochondriacal and bored, and who have improved as a result of the war. As I have mentioned elsewhere, one old lady insisted on moving up to Southeast London, as she felt she could be more useful there than in the suburbs.

Organic Factors—As a result of organic theories of shell-shock becoming discredited, I think there has been a considerable swing of the pendulum in the opposite direction, and a tendency to ignore organic factors. I have been struck by the fact that a number of patients complaining of functional disorder have been persons who, before being subject to bombing, had never previously suffered from their nerves. They did not impress me as neurotic personalities and—in confirmation of their statements—had often not consulted a doctor for some weeks after being blown up, which was consistent with their not being "neurotic." All of them gave a history of having either been knocked unconscious for a short while, or having been severely knocked about by blast. Two such cases exhibited a condition of "commotio retinae," slight oedema of the retina and optic disc, six months from repeated vivid dreams of the terrifying experience undergone. Some also exhibited mild hysterical features, but these (as explained) could be accounted for by associated psychological stress. The striking thing was that their severe, prolonged and intractable anxiety, with no gain to themselves in view of their past history, seemed incongruous; and I feel that due weight should be allowed for a psychological disturbance which perpetuates whatever biochemical changes a psychological shock leaves as its aftermath.

I have also seen a number of middle-aged patients who went through the last war quite unaffected, but who have developed anxiety and depression in this on very slight provocation, again to my mind suggesting some psychological, or possibly cerebral arterial change behind the psychological state. To those with experience of head injuries all this may seem self-evident, but among psychologists I do think the organic side frequently tends to be overlooked.

Acute Conditions—These arise in patients who are admitted shortly after undergoing

* It is interesting, in this connection, to record that I have seen only one case out of about 100 Service cases. I think the explanation is somewhat similar to that given above. The malingerer turns to an "honest-to-God" physical complaint fearing that to complain of nerves would be too near to an admission of fear.

some terrifying or horrifying experience, sometimes following a period of stress associated with lack of sleep. They are intensely emotional, tremulous, easily burst into tears, and may appear confused. In one case a patient lay on his back, staring, mumbling, calling out, and answering unintelligibly when questioned. Only when pressed did he become rational and even then relapsed immediately after. Such patients give the impression of being confused and disorientated, but as they have been too far away to suffer any blast effects, this confusion is not organic but emotional. Patients respond well to treatment.

The technique of treatment is firstly to try and get the patient to abreact by talking of his experiences. A sodium amytal or similar narcosis may facilitate this. (Sergant and Slater have advocated increasing the dose to produce unconsciousness if the patient is finding the recitation too painful.) In any case, after producing an abreaction, or even if it should fail, the patient is kept asleep from 48 to 72 hours, during which period he is given large quantities of glucose saline solution. The usual drugs employed for continuous narcosis are suitable, and I have found rectal paraldehyde very satisfactory. The immediate results of such treatment appear to be good. The patient wakes up very much quieter and, after the effects of the drug have worn off, is much refreshed. At this stage one or two talks about his problems are useful, though it may be wise to refrain from discussing the precipitating experience. The sooner such a patient gets back to normal life the better, if a conditioned anxiety is to be avoided. Whether this occurs will depend largely on his make up.

Children—The incidence of neurosis among children is fortunately very small. Very few reach hospital. I have seen only one really bad case, that of an under-developed mental defective, aged 14, who had been through many raids with little effect on him, but who was brought to hospital following the death of his small brother in a collapsed basement. This child was in an acutely emotional state of the type just described. However, my opportunities for seeing children have been small. But the superintendent of a special neurological hospital confirmed the small incidence of neurosis in children.

Mrs. Hardcastle, Organiser of the Emergency Mental Health Committee, finds that children are more apt to be affected indirectly by anxiety on the part of parents, than directly. She expressed concern as to the psychological aftermaths in children who have seen terrible sights, but as yet it is too early to predict whether serious sequelae will follow. Children are surprisingly resilient, and with suitable care one hopes that the effects will not be too bad. All told, they are surprisingly unmoved. Mrs. Hardcastle of Coventry, instances the case of one child who, after the first bad blitz on Coventry, was being driven through the ruins. With a masterly capacity for understatement, worthy of his Prime Minister, he just kept remarking: "Naughty Jerry, naughty Jerry!" She also says that children in rest centres "all too realistically play 'bombs'!"

Social factors—As already indicated these play an important role. Financial insecurity, the destruction of business, and—even more so—potential business, are all naturally apt to produce anxiety, and a consequent desire for compensation.

Worry about relatives and grief at their loss are important causes of anxiety and depression. I have heard some shocking histories, such as one man who first lost his fiancee, and then his parents, in one week, and then was in a house where everyone but himself was killed.

Predisposition — How far individuals are likely to break down under stress is an important problem, and one about which more information is urgently required, especially with regard to enlisting men to the colours. (Even if the information was available one wonders how much use medical boards would make of it. There generally appears to be no attempt to elicit the most rudimentary psychiatric information.) The main problem, of course, is not only to find positive family and past histories, or instability of one sort and another, but how to weigh them. Authorities with experience in the last war disagree on this score, some contending that "neurotics" often proved themselves valuable. Curran and Mallison produced a very interesting paper comparing the past and family histories of sailors who had psychiatric complaints with a normal control group of

wounded. They summarized their findings as follows:

"An investigation was made into the incidence of different forms of psychiatric disorder in 100 admissions to the neuropsychiatric unit of a naval auxiliary hospital.

"The proportion of breakdowns which might have been predicted by previous examination has been estimated at 39 per cent.

"The family history, past history, personality and physical findings in these cases have been compared with those of 50 surgical patients in an attempt to discover which factors should be regarded as the most useful indications of instability. It appears that the proportion of positive past histories was high among the predictable cases, and of positive family histories among the unpredictable cases.

"Valuable manpower might be economized by getting a full history from recruits, arranging for a psychiatrist to interview unsatisfactory recruits in the training establishments, excluding mentally defective men by the use of group tests, and instituting tests of personal aptitude. Some patients who have broken down might give good service on shore duty if not fit for full duty. Others might be drafted to rehabilitation camps, or be given further treatment at psychiatric clinics, which should be made available for invalided naval ratings. A report should be sent to the labour exchange with patients invalided out of the Service, so that they may be found appropriate work."

Compensation—As a result of the enormous sums spent on pensions for "shell shock" and "neurasthenia" in ex-Servicemen after the last war, the authorities have taken up the stand of "never again." War pensions can theoretically only be secured for *"commotional"* as apposed to *"emotional"* disturbance arising from enemy action, though they receive support in the latter cases, and their dependants allowances if they need in-patient treatment. Actually, partly as a result of the weird and wonderful certificates which those quite unversed in psychiatry will issue, e.g., "suffering from bomb-shock," and owing to a Nelsonian blind eye being turned to allowances paid for a short while, some payments are made. But if they are to be continued they are subject to strict review, and here the question of the role played by constitutional versus precipitating factors, is a perpetual difficulty.

The greatest difficulty, of course, arises because one is assessing entirely subjective symptoms. It is necessary to decide how far these are real as opposed to suspicions, and how far financial gain may prevent a patient's making the necessary effort to overcome his disorder. But most patients I have seen have struck me as suffering genuine anxiety with little added hysteria, and who are in consequence to some degree disabled. There is a further problem in this war. As industrial areas have come into the front line, this limits the scope for employment of those whose nerves have gone. Therefore, the patient with a psychiatric disorder finds himself at a particularly severe disadvantage. In spite of all this, the principle of paying a man as long as he has symptoms for which there is no objective corroboration is an undesirable one. It appears to me that the solution might be along the lines of paying a lump sum. The question is too large to be discussed here, but it is one which would repay considerable attention, for the ideal solution has yet to be found.

In this article I have mainly confined myself to the problems of civilians, as I feel these new problems would be of the greatest interest to the American reader. The first part of the paper was written rather earlier than the rest, and in any case it should be understood that the paper as a whole consists of a number of cross-sections. Events move rapidly, and impressions and findings are apt to differ from time to time.

I have referred little to Service cases, about which considerable literature exists. (In any case, clinically they show the same pictures as those described here, and treatment is along the same lines). I also wish to make it clear that this is far from being an exhaustive description, but I have succeeded, I hope, in indicating some salient features of the psychological effects of war on the civilian.

I am very much indebted to Mr. Tom Harrisson, who has gone to considerable trouble to furnish me with much valuable information collected by "Mass Observation." My references include papers not quoted in this article, but a complete list of the main wartime psychiatric literature may prove useful to the reader.

The Gingival Changes Produced by Dilantin Sodium*

LEON J. ROBINSON, M.D.
Palmer, Massachusetts

Dr. Robinson is Senior Physician at Monson State Hospital. He received his M.D. degree from Boston University School of Medicine in 1934. Upon graduation, he served as House Officer for 18 months at the Boston City Hospital and, following this appointment, as Resident and Assistant Physician at the Monson State Hospital. He is a Diplomate of the National Board of Medical Examiners; a member of the International League Against Epilepsy, and the Massachusetts Society for Research in Psychiatry. He is Chairman of the Publication Committee of this latter organization and also a Diplomate in Psychiatry and Neurology of the American Board of Psychiatry and Neurology.

In the treatment of epilepsy, dilantin sodium (sodium diphenyl hydantoinate) has continued to establish itself as a valuable therapeutic agent. First introduced clinically as an anti-convulsant by Merritt and Putnam,[1] it has been reported upon favorably by many investigators. Its toxic effects were also early recorded, Merritt and Putnam[1] pointing out in their first clinical report that these might include dizziness, ataxia, tremors, blurring of vision, diplopia, slight nausea, dermatitis or purpura.

Kimball[2] first called attention to the gum changes occurring in many patients receiving dilantin sodium. He made clear that the changes did not contraindicate continuation of medication which had brought about improvement in seizures, mental state, personality and general health in all but 22.5 per cent of his cases. Of his 119 cases re-examined for hyperplasia of the gums, 68 showed varying degrees of hyperplasia and of the latter, 17 patients had advanced hyperplasia. He found that 57 per cent of patients on dilantin sodium for two months or more showed changes in the gums to some degree, but some patients who had taken the drug for eleven months showed no change. Some had a sore mouth and tender gums for a short period starting about two weeks after the beginning of therapy. Those showing definite beginning hyperplasia were designated one plus, those with rather advanced hyperplasia two plus, and those with extreme hyperplasia three plus. In only 4 cases was the advanced hyperplasia associated with soreness and petechial hemorrhages on pressure and in each of these there was an associated gingivitis. No patient who had employed good oral hygiene had any suggestion of sponginess or bleeding. The others showed a smooth firm hyperplasia that did not bleed easily and was not particularly tender. Kimball reported definite lowering of the ascorbic acid levels in blood serum paralleling the severity of gum hyperplasia. According to Kimball,[2] where the gum was normal, the blood serum ascorbic acid in mgm. per 100 cc. measured 1.14; where the gum was one plus, it measured 0.58; where two plus, it measured 0.55; where three plus, it measured 0.47. He did not find the usual symptoms of severe and prolonged scurvy such as purpura, sore joints and extreme weakness. His patients often noted dizziness, ataxia and unusual spells of weakness. With therapy of 300 mg. of ascorbic acid given daily, the condition of the gums was only slightly improved in three weeks and the blood ascorbic acid was about 1/10 of normal.

Gruhzit[3] was unable to produce a significant reduction of blood ascorbic acid in animals despite continued administration of dilantin sodium.

In Merritt and Putnam's report on the toxic effects of dilantin[4] hypertrophy of the gums was noted in 6 per cent of their patients and practically exclusively in children or young adults. It was not noted by the patient and was either found on examination or reported by the parents because they or their dentists had noted it. These authors observed that the hypertrophy of the gums was different from that seen in patients with scurvy in that it was firm, painless and free of bleeding and unaccompanied by other symptoms of scurvy.

Merritt and Foster[5] found that 40 of 182 patients (22%) who were receiving dilantin sodium therapy had some degree of gingival hyperplasia but in only 5 patients (3%) was the swelling severe enough to cause discomfort. Their determinations of the plasma vitamin C content failed to show any rela-

* Presented before the Massachusetts Society for Research in Psychiatry, Palmer, Massachusetts, April 15, 1941.

tionship between the vitamin C content and the gum changes. The average content of 0.44 mg. of vitamin C per 100 cc. was exactly the same in both patients with and without the gingival sign. Furthermore, they determined the plasma vitamin C content in epileptic patients not receiving dilantin therapy, 31 of whom received no anti-convulsant treatment and 44 of whom received phenobarbital, and found that the vitamin C content was no different from that in the dilantin treated group. Even continued administration of dilantin sodium over a period of two years failed to produce an appreciable difference in the vitamin C content as compared to vitamin C level in patients not receiving dilantin sodium. The vitamin C level depended solely upon the amount of vitamin C containing food ingested. They placed their patients on a high enough vitamin C containing diet to raise the average ascorbic acid level from 0.22 to 1.12 mg. per 100 cc. without appreciably affecting the gum swelling.

Other investigators have reported variable findings from complete absence of gum changes to as high an incidence as 50 per cent. Those who have found no gum changes in patients receiving dilantin, but who were nevertheless aware of this possibility, were Phillips[6] treating 12 patients; Frost[7] treating 9 patients; and Williams[8] treating 91 patients. Weaver and his colleagues[9] made no mention of the gum condition in their 14 patients but neither did they indicate that they were acquainted with the possibility of such changes. Blair and his associates[10] treating 75 patients found but 2 with gum changes bearing any similarity to Kimball's[2] description despite examination by a qualified dentist. Steel and Smith[11] treating 20 patients found 3 with gum changes. Hodgson and Reese[12] found that of 44 patients, 3 showed gum hyperplasia; McCarton and Carson[13] of their 20 cases reported one with hyperplasia. Pratt[14] stated that although 50 per cent of 52 patients on dilantin showed mild hyperplasia, a similar incidence of such change was discovered by a qualified dentist even in patients not receiving dilantin therapy. Robinson and Osgood[15] in recording their results with dilantin sodium recorded the pres-

Stage 2

Stage 3

ence of gingival changes as one of the toxic effects.

Observations of Present Study

The gingival changes accompanying dilantin sodium occur under definite conditions and with a fairly characteristic progression in the appearance of the gingivae. The age of the individual is an important factor. The highest incidence of gum changes is found in the younger age group. (Thus children are more susceptible to this complication and should be more frequently observed for its occurrence). The gingival alterations are a local reaction unaccompanied by any demonstrable constitutional aberrations. Another important factor is the presence of normal gum tissue, as areas in which there is pyorrhea alveolaris do not show the alterations which adjoining areas free of Rigg's disease present. Another striking fact is the absence of dilantin gum changes in edentia or areas where even a single tooth is absent.

As a rule there is a constant and characteristic progression of the changes so that it is possible to recognize five stages in the development of the process (see illustrations). Early, they are best seen on the anterior surface of the gums though present on anterior and posterior surfaces, and are primarily limited to the areas of the incisor and canine teeth.

The first stage is characterized by a more intense redness of the gum at the dental margin. This is a darker color than the contrasting pink of the adjoining normal gum and forms a narrow horizontal band. Often it is purplish red. This narrow horizontal dark red band adjacent to the tooth is not raised above the surface in the first stage. There is no bleeding or pain.

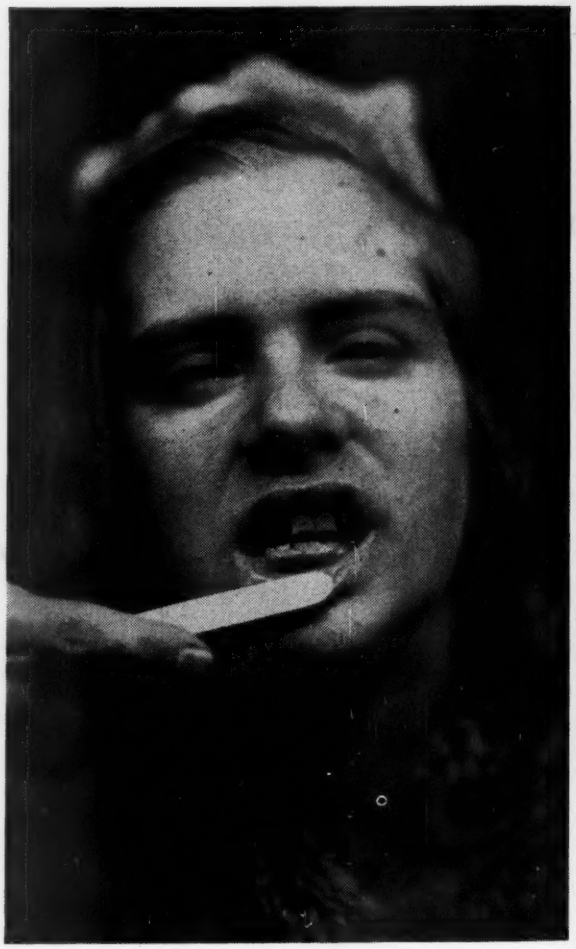
In the second stage there is added an elevation of the interdental areas of the gum. The interdental portions of the gum are raised above the surface of the adjoining gum and present the appearance of being studded with small islands of firm redundant gum tissue. There is neither bleeding nor pain.

In the next or third stage the marginal gum areas become raised, and in the interdental spaces redundant tissue extends in a distal direction partly occluding the space between the teeth. The gums do not bleed and there is no pain.

The fourth stage shows a more generalized overgrowth of both the marginal and interdental gum tissue. The entire areas involved present a heaped up appearance. The color may be either red, as in the early stages, or pale as though the vascular bed is compressed by excessive expanding gingival tissue. In this stage also the gum presents a fungating appearance and may bleed if traumatized even slightly by food or finger.

In the fifth stage there is an extension of the redundant tissue over the anterior and posterior surfaces of the teeth. Finally the teeth may be entirely buried so that in place of the biting edge of the teeth one sees only a mass of gum pulp. Such a condition makes it both difficult and painful to chew, in contrast to the previous four stages which are painless.

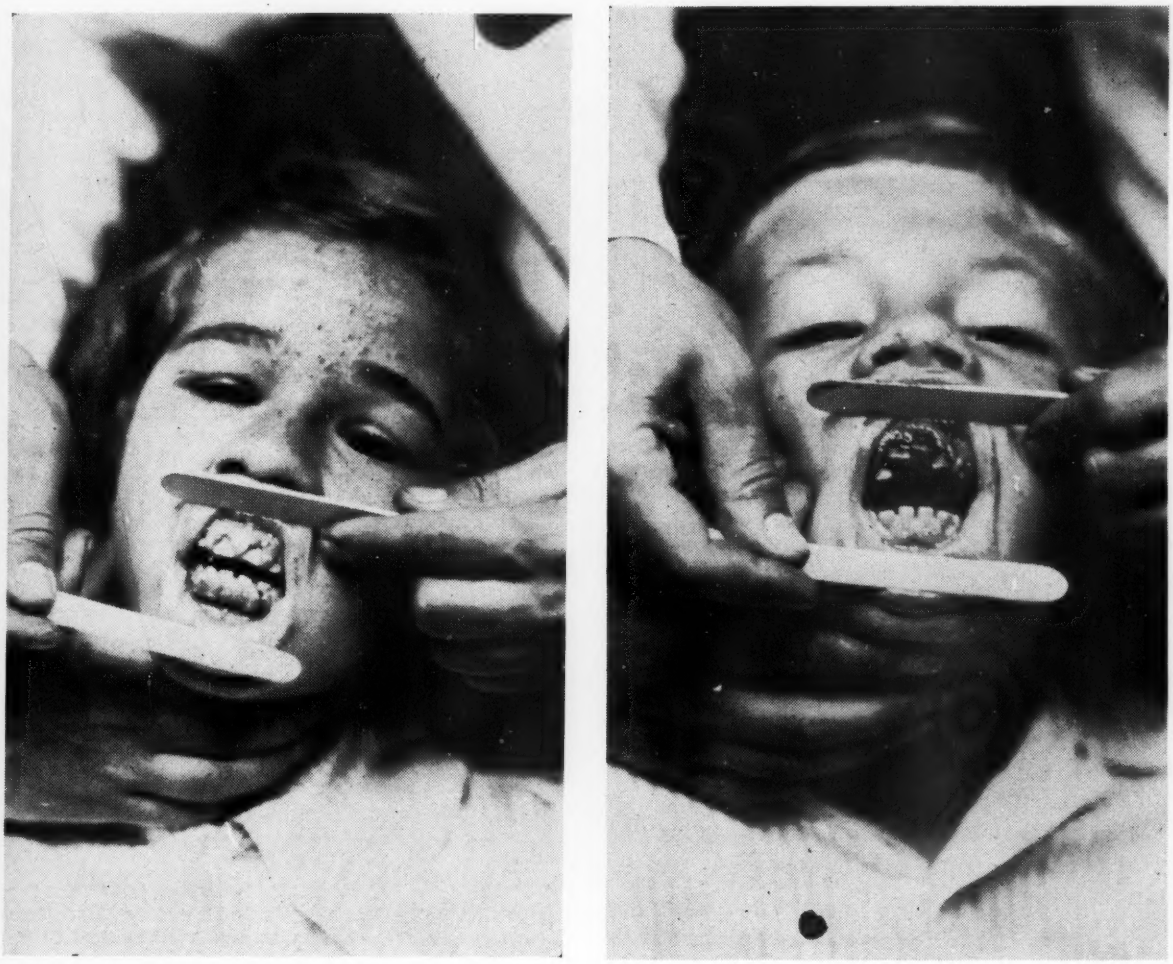
The gum changes show no definite time of onset. They may appear as early as the second week under dilantin therapy or they



Stage 4

may appear after several months despite continuation of the same previously well tolerated dilantin dosage. Again there is no predictable course in its regression. The gingival changes may recede although medication is unchanged. In practically every case, withdrawal of the dilantin medication results in the gradual disappearance of the gingival changes over the period of several weeks to three months. However, exceptionally, the gingival changes may require a longer time. In one case in our series the marked gingival alterations have persisted, with only mild regression, despite complete withdrawal of dilantin over a period of 16 months.

In tabulating the amount of dilantin sodium administered it became apparent that the gingival changes are not dependent on the size of the dosage but rather on the sensitivity of the individual. Many of our patients on the higher dosages of 7½ to 13½ grains (0.5-0.9 grams) daily for over a year showed no changes whereas others who were susceptible to such change presented alteration of the gums as a result of receiving 3 grains (0.2 grams) for only three weeks. The fact that changes occur does not demand reduction or omission provided the gums do not progress beyond the third stage outlined above, provided the patient is closely observed at intervals of two to four weeks, and provided, of course, the dilantin sodium produces a satisfactory anti-convulsant effect. A person who has at one time shown gum changes may receive another trial of dilantin therapy at a later time. For example, a child of ten showing such changes may not do so a year later. In our series of cases, recurrence of the condition on renewal of dilantin sodium was prone to occur when the prev-



Stage 5 *Stage 5*

Gingival changes produced by Dilantin Sodium. A hand lens improves the details. Note the nodular appearance as well as the overgrowth in Stage 5.

ious gingival reaction had been marked.

The statistical incidence of gum changes was 19 per cent of our group comprising 143 patients. For the most part they were mild changes and often disappeared without the use of special measures. In only 5 per cent of the cases were there the more marked changes such as occur in the second to fifth stage. In only one case did the marked changes persist despite the withdrawal of dilantin medication. As has already been noted there was frequently a regression of the pathological process despite continuation of dilantin therapy. It should be noted that although the younger age group was more prone to develop gum changes it was not unusual to find them in young adults. Both sexes showed equal susceptibility to the gingival effects of dilantin sodium.

The manner in which dilantin sodium produces the gingival changes is unknown. Correctional measures have yet to be uncovered. Because Kimball[2] found decreased blood ascorbic acid in association with the gum changes he administered 300 mgm. of ascorbic acid daily but with little benefit at the end of 3 weeks. None of our cases showed any clinical evidence of scurvy such as petechial increase with the tourniquet test, pretibial tenderness nor osseous roentgen changes consistent with scurvy. We, however, gave patients with gingival changes, 450 mgm. of cevitamic acid daily for 6 months without preventing or alleviating the gingival changes. Because of Ziskin's favorable report on estrogenic therapy (progynon B—injectable) in some gingival conditions such as hypertrophic, proliferative and desquamative gingivitis,[16] based upon his finding that these substances caused epithelial hyperplasia and hyperkeratinization of the gingivae and oral mucous membranes similar to these changes in nasal and vaginal mucosa,[17] I experimentally administered progynon DH (Schering) orally using 600 units daily for three months without noting any prophylactic or correctional effect upon gingival changes from dilantin sodium. As we have already pointed out, the only certainly effective means of insuring regression of gum changes consisted in the withdrawal of dilantin sodium.

Table I presents some of the facts relating to the gingival changes observed by several investigators, and includes the present study.

Summary

The therapeutic use of dilantin sodium as an anti-convulsant agent was accompanied by gingival changes in 19 per cent of children and young adults of both sexes, but was marked in only 5 per cent.

The gingival appearance ranged from redness to varying degrees of redundant overgrowth, unaccompanied by local or general symptoms, and seen only in areas where there had been normal gum tissue and where teeth were still present. There were no clinical signs of scurvy.

The changes bore no relation to the dosage of dilantin sodium, and occurred at any time during its administration, but not earlier than the second week.

Gradual regression of the gingival changes followed withdrawal of the drug, but could not be induced by the use of ascorbic acid 450 mgm. daily, or progynon DH 600 units daily. Only the rarer extensive gingival changes necessitated discontinuance of anti-convulsant therapy with dilantin sodium.

Monson State Hospital.

Note

Subsequent to completion of this study, D. E. Ziskin, L. R. Stowe and E. V. Zegarelli *(Arch. Neurol. and Psych.*, 46: 897-907, November, 1941) described the pathology of the gingival changes as a hyperplasia, with little keratin change, epithelial proliferation and marked connective tissue proliferation with many fibroblasts and masses of inflammatory exudate. X-ray might reveal loss of interseptal bone. Surgical excision of the gum was temporarily effective, but was followed by recurrence. Interdental gum massage was therapeutically effective.

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TABLE I

REPORTED GINGIVAL CHANGES PRODUCED BY DILANTIN SODIUM

Observer No. Cases	*Incidence*	*Age*	*Sex*	*Time Onset*	*Gingival Appearance*	*Local Symptoms*	*General Symptoms*	*Treatment Tried*	*Dilantin Discontinued*	*Comment*
Kimball 119	Total 57 0/0 Marked 14 0/0	Children Young Adults	Either	Any Time Earliest 2 Weeks	Hyperplasia 1 to 3 Plus. No Relation to Dose.	Sore Mouth and Tender Gums in Some	Often Dizziness Ataxia Weakness	Ascorbic Acid 300 mg. Daily, no Appreciable Effect	No	Reported Decrease of Blood Serum Ascorbic Acid Parallel to Severity Gum Changes. No Other Signs of Scurvy
Merritt and Putnam 350+	6 0/0	Children Young Adults	Either	No Mention	Firm, non-Bleeding Hypertrophy No Classification of Degree. No Mention Relation to Dose.	No	Not Usual	No	No Mention	
Robinson L. J. 143	Total 19 0/0 Marked 5 0/0	Children Young Adults	Either	Any Time Earliest 2 Weeks	Five Stages: Redness to Marked Gingival Overgrowth. No Bleeding Without Trauma. No Relation to Dose.	Rarely Unless Interference Chewing	No	Ascorbic Acid 450 mg. Daily, ineffective. Progynon DH 600 Unit Daily, ineffective.	Only if Extensive Reaction	No Clinical or Roentgenologic Signs of Scurvy

Reports of Incidence: Blair Et Al (75) 2 1/2 0/0; Steel and Smith (20) 15 0/0; Hodgson and Reese (44) 7 0/0; McCarton and Carson (20) 5 0/0; Merritt and Foster (182) 22 0/0.

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Book Review

ENCEPHALITIS, by Josephine B. Neal, A.B., M.D., Sc.D., F.A.C.P., Associate Director, Bureau of Laboratories, Dept. of Health, New York; Clinical Professor of Neurology, College of Physicians and Surgeons, Columbia University. Pub. by Grune and Stratton, New York, 1942. Pp. 521, plus 16 plates and index.

This book, a publication of the "William J. Matheson Commission for Encephalitis Research," is edited by Josephine B. Neal, M.D. It consists of a series of papers by various members of this group, and each paper is presented as an individual production.

The first section contains an excellent historical survey of the development of the present knowledge of encephalitis and a brief discussion of the various forms now recognized. The differential diagnostic survey is complete.

The chapter on the epidemiology of epidemic encephalitis is brief and gives little information not present in the first section.

The portion devoted to the neurological complications following infections and vaccinations consists of a thorough survey of the literature, but contributes nothing new to the knowledge of these conditions.

Chapter IV discusses the clinical course of epidemic encephalitis. The discussion is thorough and fairly well organized. The discussion of late sequelae is excellent.

The section on the treatment of epidemic encephalitis presents interesting data on the use of a herpes virus vaccine. The improvement noted from its use is hard to reconcile with the evidence presented to show that the herpes virus is not the etiologic agent in this disorder. The author's reasons for limiting the therapeutic investigation to vaccines of various types appear arbitrary.

The discussion of the treatment of the chronic phase of encephalitis contains nothing new, but is a reasonably complete survey of the established methods.

The chapter on the surgical treatment of the postencephalitic syndrome is a complete and well-written survey of the present status of the surgical management of the late tremors and spasticity.

The section on the psychiatric sequelae of encephalitis adds nothing but bulk to the book.

In contrast, the chapter on Postencephalitic Behavior Disorders in Childhood contains a careful survey of the literature and an excellent description and discussion of the symptoms.

The section on pathology is detailed, factual and clearly presented. It is by far the best portion of the book.

This report of the commission will not bring about any revolutionary change in our concepts of the various encephalitic illnesses, nor greatly increase the effectiveness of our treatment. The book can be recommended as a collection in one volume of the important literature on all phases of the various types of encephalitis, and as such deserves a place in every medical library.

Jack Ewalt, M.D.
Galveston, Texas